ON MULTIPLE SEROSITIS—THE ASSOCIATION OF CHRONIC OBLITERATIVE PERICARDITIS WITH ASCITES, WITH PARTICULAR REFERENCE TO THE "PERICARDITIC PSEUDOCIRRHOSIS OF THE LIVER" OF PICK AND THE "ICED LIVER" (ZUCKERGUSSLEBER) OF CURSCHMANN.[1]

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For purposes of study, cases of chronic adhesive or obliterative pericarditis may be divided into three classes: first, cases in which the pericarditis runs its course entirely devoid of symptoms and constitutes merely an accidental though interesting finding at the necropsy; second, cases in which, sooner or later, symptoms of disturbed or impeded cardiac action manifest themselves, and in which, from the physical signs, the obliterative pericarditis is more or less susceptible of diagnosis; and, third, cases characterized clinically by marked ascites, with little or no œdema of the legs, cases in which the diagnosis of cirrhosis of the liver is usually made and which at the necropsy reveal chronic obliterative pericarditis and certain morbid changes in other tissues and organs—notably pleuritis, peritonitis, perihepatitis, nutmeg liver, red atrophy of the liver, cirrhosis of the liver, etc. It is to the third class of cases that I wish to direct attention.

In the majority of cases when chronic adhesive or obliterative pericarditis gives rise to symptoms, the symptoms that arise are those of failing cardiac compensation—palpitation of the heart, dyspnœa, cough, cyanosis, generalized œdema, effusions into the several serous cavities of the body, etc. On the contrary, in the cases that simulate cirrhosis of the liver, clinically, the first symptoms are sensations of fulness, oppression, and weight in the upper abdomen, or there may be apparently a sudden onset with acute pain; the abdomen increases in size and may attain very large dimensions—due evidently to ascites that may necessitate repeated tappings (Rumpf's patient was tapped 301 times).

With increase in the amount of the ascites the abdominal walls become tense and painful, and the other subjective symptoms become markedly aggravated. There may be slight and transitory jaundice. Later œdema of the legs may develop. In a few cases œdema of the legs comes on early; it may remain but a short time, disappear, and not recur until shortly before the death of the patient. In rare cases it persists; but in the class of cases to which I refer it is always inconspicuous when contrasted with the excessive and constantly recurring ascites, and it scarcely attracts attention.

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[1] Read, by invitation, at a meeting of the Johns Hopkins Hospital Medical Society, Baltimore, and at a meeting of the College of Physicians of Philadelphia.

This symptom complex of obliterative pericarditis is of uncommon occurrence. While by no means unknown to the older writers, it attracted but little attention, and it was not until the communication of Pick, in 1896, that it became the subject of much discussion. Even at the present time it is overlooked by a number of systematic writers on diseases of the heart and of the liver, and a search through a number of recent text-books on medicine reveals little or no mention of it.

In the present state of our knowledge, it is to some extent a matter of personal opinion what cases should be included in the group of cases under discussion. From the point of view that I hope to elaborate, the earliest recorded case that I should include within this group was reported by van Deen in 1846. Subsequently cases were reported by Frerichs, Feierabend, Hambursin, Weiss, Vierordt, Tissier, Hirschler, Weinberg, Mott, Variot, Riedel, Henoch, Harris, Broadbent, and Dickinson. In 1884 Curschmann described his now well-known "zuckergussleber," or chronic hyperplastic perihepatitis—a disease characterized by a slow, insidious, and intermittent course, and persistent ascites, but without connective tissue hyperplasia in the liver. Although usually described as the "zuckergussleber" of Curschmann, this chronic hyperplastic perihepatitis was well known to a number of the earlier writers, being described by Hilton Fagge, in 1875, by Wilks and Moxon, in 1875, by Murchison, in 1868, by Bamberger in 1864, and by Rokitansky, in 1842. In 1895 a paper on the same subject, with the report of a case, was published by Rumpf. In 1896 Pick, reporting three cases, directed attention to the symptom complex simulating that of cirrhosis of the liver, but found at the necropsy to be due to obliterative pericarditis and associated changes in other tissues.

Pick pointed out that the recurring ascites with almost entire absence of œdema of the legs, the enlarged, firm, and somewhat irregular liver, and absent or slight jaundice, strongly suggested cirrhosis of the liver, and that the obliterative pericarditis usually escaped clinical recognition—although he was enabled to make the correct diagnosis during life in the third of his reported cases. Pick suggested for these cases the designation "pericarditic pseudocirrhosis of the liver"—a designation that has since found favor with some medical writers, and he endeavored to establish an etiological relationship between the obliterative pericarditis, the enlarged liver, and the ascites. He stated that as a consequence of the pericarditis, which he looked upon as the primary disease, disturbances of circulation, with consecutive connective tissue hyperplasia in the liver, occurred, and that as a result of these ascites developed. In this class of cases he included the cases of "zuckergussleber" previously reported by Curschmann and by Rumpf, Following Pick's paper, the subject attracted considerable attention, reports of a number of cases appeared, discussions relating to the nature and the sequence of the morbid processes arose, and the interpretations of the lesions were many. Mention may be made of the papers by Heidemann, Hübler, Siegert, Werbatus, Nachod, Taylor, Ewart, Diemer, Schmaltz and Weber, Rose, Eisenmenger, Pick, Giradeau, Rosenbach, Hutinel, Türk, and others.

A disease that is identical with that under discussion has been well recognized in Italy, being known in some quarters as Concato's disease, polyorrhomenitis, or polyserositis. In 1891 Picchini published an extended report of the disease—basing his remarks upon personal observations and a critical study of the cases reported in the literature. Interesting papers were published by Cantu in 1894, and by Villani in 1895.

In 1898 Hager, a German author, writing on the subject, reviewed critically the Italian literature. Since the publication of Pick's paper, papers by Schupfer, Bozzolo, Galvagni, Patella, Caporali, and others have appeared. In France the disease is sometimes known as perivisceritis.

The only reports of cases by American writers that I have been able to find are those by Osler, in 1896, by Cabot, in 1898, and by Herrick, in 1902.

The subject has not been entirely neglected by systematic writers. Thus, in 1887, Rosenbach stated that pericardial obliteration is sometimes associated with extensive fibrous perihepatitis, enlargement of the liver, and ascites, "so that one might presume that enlargement of the liver, ascites, and pericardial synechiæ stand in causal relationship the one to the other." He refers to the subject also in his book on diseases of the heart. In 1890 Strümpell wrote that in cases of pericardial obliteration, he, as well as others, had sometimes observed marked ascites (at times associated with hydrothorax) without coincident œdema of the legs. In the latest edition of his text-book on medicine, referring to the subject, he states that all the cases presenting the symptom complex under discussion are not of one nature. In 1891 Leube wrote that pericardial obliteration sometimes gives rise to swelling of the liver and consequent ascites. In 1894 Schrötter wrote that pericardial obliteration sometimes gives rise to ascites before the occurrence of general anasarca. The subject was referred to also by Osler, in the second edition of his text-book on *Practice of Medicine*, in 1895, by Quincke and Hoppe-Seyler, in 1899, and by Roberts, in 1900.

The subject may be illustrated by the report of the subjoined case. For the clinical notes, I am very much indebted to Dr. James C. Wilson, physician-in-chief, and to Dr. John B. Deaver, surgeon-in-chief, to the German Hospital.

Henry S., single, white male, aged thirty-one years, a native of Sweden, and a clerk by occupation, was admitted to the medical wards of the German Hospital, May 31, 1898, and the following history elicited:

He is the only child of his parents, both of whom are living and well. He has no knowledge of his having had any of the diseases of childhood. From an early age, however, he had been subject to convulsive seizures attended with unconsciousness. It is reported that his mother was similarly affected. The spells, as they occurred in the patient, came on at irregular intervals and lasted for from several minutes to about an hour. When about sixteen years of age they increased very much in frequency. At times he had a premonition of their oncoming. During some of the lighter attacks he did not lose consciousness entirely, that is, he was more or less aware of what was transpiring about him, but he was unable to speak. When about eight years of age he had severe pains in both groins and in the upper part of both thighs. At times these were so severe as to prevent his lying down.

At about the same time he experienced paræsthetic phenomena in the rectum—he felt as though there was something creeping about in the rectum. At the age of eighteen years he acquired gonorrhœa. One year later he had a severe attack of rheumatism that lasted about three months. Since then he has had a number of minor attacks of rheumatism, but none was severe enough to disable him. In 1890 he was treated in Bellevue Hospital, New York, for what he states was malaria. (A note from the Medical Superintendent of Bellevue Hospital, under date of March 12, 1901, states that the patient was treated in the hospital for malaria, from July 13 to 18, 1890. No details of his condition at that time are available.) For a short time during 1892 the patient was confined to an asylum. He had suffered a sunstroke and had become, he states, maniacal at times. Since then he has been troubled with disordered digestion, or "stomach trouble," as he expresses it. Although he is said to have been jaundiced at times, the most obtrusive symptom has been periodic pains in the right half of the abdomen just above the right iliac fossa. These occurred at first at intervals of about a month, later at intervals of two months or more.

During April, 1898, the patient was in the Jefferson Medical College Hospital for about two weeks. (Through the courtesy of Dr. H. A. Hare, I have ascertained that while in the hospital the patient was slightly jaundiced, and at times delirious, that he had several convulsions, and that although he complained of severe pain in the right upper abdominal quadrant and of tenderness over the region of the gall-bladder, careful examination under ether failed to reveal any tumor in the region of the gall-bladder or other evidences of gallstones. The liver was slightly enlarged, and the superficial veins, especially those on the right side of the abdomen and the inner side of the right thigh were distended. The urine at this time was acid in reaction, had a specific gravity of 1025, contained 3.2 per cent. of urea, and neither sugar nor albumin. Examination of the blood revealed: erythrocytes, 5,200,000; leucocytes, 24,000; and hæmoglobin, 73 per cent. A differential count of the leucocytes revealed: small lymphocytes, 4 per cent.; large lymphocytes, 5 per cent.; transitional leucocytes, 4.9 per cent.; polynuclear leucocytes, 85.1 per cent.; and eosinophiles, 1 per cent.) Shortly after leaving the Jefferson Medical College Hospital the patient's abdomen began to enlarge, the pains in the right side of the abdomen became more severe and more frequent, so that he was incapacitated for work. He was admitted to the German Hospital complaining of severe pain and tenderness in the right upper abdominal quadrant, especially over the region of the gall-bladder, which was aggravated by deep inspiration; of pain over the pubes after micturition; at times of general pain in the right side of the abdomen; sharp cutting pains about three inches within the anus; attacks of vertigo; spots before the eyes; and of constipation of three years' duration. He smokes moderately, and formerly he consumed considerable quantities of alcohol.

On examination the only noteworthy sign was marked distention of the abdomen, due evidently to ascites. His liver dulness extended upward to the third rib. On June 2 the patient's abdomen was tapped, and 4400 c.c. of clear amber fluid was withdrawn. The fluid had a specific gravity of 1015, and contained a few erythrocytes and many leucocytes. The tapping relieved the patient very much, and afterward the liver dulness was found to be normal in area. There was marked tenderness over the region of the gall-bladder. Examination of the blood, on June 3, revealed the following: hæmoglobin, 82 per cent.; erythrocytes, 3,930,000, and leucocytes, 7300. A differential count of the leucocytes revealed the following: small lymphocytes, 11 per cent.; large lymphocytes, 9 per cent.; transitionals, 3 per cent.; polynuclear neutrophiles, 76.5 per cent., and eosinophiles, 0.5 per cent. On June 13 it was noted that the ascites was gradually reaccumulating, and the pains were becoming more frequent and more marked. On June 22 the patient was again tapped and 6700 c.c. of clear amber fluid was withdrawn. The fluid had a specific gravity of 1018, and contained a few erythrocytes and many leucocytes. On July 2 it was noted that the pain in the region of the gall-bladder had been absent since the second tapping. On this date the patient's feet were found to be slightly swollen—the first swelling of the feet since he had been in the hospital. (Upon inquiry he admitted that they had been slightly swollen on one previous occasion.) On July 9 the patient felt better than he had during the previous six months, and he was free from pain. On August 9, feeling well, he left the hospital of his own accord. His feet were not swollen at all, and he suffered no pain. His abdomen, however, contained considerable fluid, and was greater in circumference than it was when he entered the hospital. On the second day after his admission to the hospital his temperature was 102° F., and on several occasions subsequently it reached 100° F. Otherwise there was no fever. His pulse was always low: on several occasions it was 80 per minute; usually it was between 60 and 68; and sometimes it was as low as 48 per minute. His urine was normal.

The patient was readmitted to the German Hospital, June 23, 1899, complaining of marked distention of the abdomen, pains in the region of the liver, at times of sharp shooting pain in the perineum, dyspnœa, slight pretibial œdema, diminution in the amount of urine voided daily, vertigo, and spots before his eyes. He stated that since he left the hospital his abdomen had been tapped twice. Inasmuch as the clinical diagnosis was cirrhosis of the liver, operation for the relief of the condition was proposed and acceded to by the patient. The operation was performed by Dr. John B. Deaver, July 3, 1899. In brief this consisted in irritating the peritoneum and stitching the omentum to the parietal peritoneum. The patient survived the operation four days, dying July 7, 1899.

The following are the notes of the necropsy performed by myself, July 8, 1899:

The body is that of a well-developed, fairly well-nourished male subject, with good bony development, good musculature, and moderate panniculus adiposus. There is post-mortem lividity in the dependent portions of the body; the skin is slightly jaundiced; there is no œdema of the extremities. In the median line of the abdomen there is a wound, about 16 cm. in length, apparently healing by first intention. The left pleural cavity is entirely obliterated by firm adhesions—the two layers of the pleura being dull, cloudy, whitish, and thickened. The left lung is about normal in size, and mottled grayish-black in color. Its air content is normal, its consistency firm, and it shows some hypostatic congestion in the dependent portions. Its section surface is smooth, moist, glistening, dark reddish in color, and exudes a quantity of frothy, blood-stained fluid. The lung crepitates throughout, and contains no tubercles. The bronchi are normal in size, they contain a small amount of mucus, and their mucous membrane is pale. The pulmonary vessels and the peribronchial lymph glands appear normal. The right pleural cavity is entirely obliterated by firm adhesions—the two layers of the pleura being dull, cloudy, whitish, and thickened. The right lung presents appearances similar to its fellow. The pericardium is firmly adherent to the anterior chest wall and the adjoining mediastinal tissues. The pericardial cavity is entirely obliterated, and such is the firmness of the adhesions that the two layers of the pericardium nowhere can be distinguished. In addition the pericardium is the seat of extensive calcareous infiltration, a large section of the heart being encased in a hard calcareous sheath. This calcareous sheath implicates the pericardium of the right ventricle, about one-half of the anterior and under surface of the right auricle, the left edge of the heart, the lowermost two-thirds of the left ventricle, the apex, and the diaphragmatic surface of the heart. This calcareous sheath appears to be made up of a single calcareous plate and not of a series of disunited plates. The pericardium of the left auricle, though firmly united with the adjacent parietal pericardium, is not the seat of calcareous infiltration. The several cavities of the heart appear to be normal in size, and they contain a moderate amount of dark, reddish-brown, coagulated blood. The mitral orifice admits two fingers, the tricuspid orifice, three fingers. The mitral and tricuspid valve leaflets and the associated chordæ tendineæ are slightly thickened. The aortic and pulmonary valve leaflets are normal. Presumably all the valves are competent. The left ventricle (including the calcareous pericardium) measures 12 to 14 mm. in thickness, the right ventricle 5 to 8 mm., the left auricle 2 to 4 mm., and the right auricle 2 to 4 mm. The heart muscle is reddish-brown in color and firm in consistency. The coronary arteries and the aorta are normal. The heart and pericardium weigh 470 grammes. The omentum is normal in size and shape. The entire peritoneum is dull, lustreless, somewhat thickened, and intensely hemorrhagic. The coils of intestine are united to one another and to the other intra-abdominal organs by moderately firm, though friable, fibrinous adhesions. In some places an exudate may be stripped off in thin layers. The peritoneum contains also about 50 c.c. of serosanguinolent fluid (drainage had been provided at the time of the operation). The liver is firmly united to the under surface of the diaphragm by means of dense and firm adhesions, especially for a distance of about 6 cm. on both sides of the suspensory ligament. The capsule of the liver is thickened throughout, but especially on its upper surface, where in some places it is 4 mm. thick. In addition, on the capsule of the liver there is some fresh fibrinous exudate that may be stripped off; this is present especially on the under surface of the liver and for a short distance on the upper surface. The liver measures 22 cm. in length, 14 cm. in breadth, and 9 cm. in thickness. It weighs 1460 grammes. Its shape is normal, its consistency somewhat increased, its elasticity somewhat diminished, its surface somewhat irregular, and its edges rounded and hard. Its section surface is dull, moist, slightly granular, opaque, and light reddish-brown in color. The liver lobules are distinct, and there appears to be a slight increase in the connective tissue of the organ. The gall-bladder is normal in size and contains fluid bile. Its mucous membrane and wall are normal. The cystic, hepatic, and common bile-ducts are patulous. The spleen measures 10 cm. in length, 8 cm. in width, and 5 cm. in thickness. It is normal in shape and weighs 210 grammes. It is firm in consistency, and its capsule is slightly thickened and covered also with some fresh fibrinous exudate. The pulp is normal, the follicles rather distinct, and the trabeculæ slightly increased. The right kidney measures 11 cm. in length, 6 cm. in width, and 3 cm. in thickness. It is normal in shape and weighs 160 grammes. Its capsule strips readily, its surface is smooth, its color dark reddish-brown, and its consistency somewhat increased. The cortex is 7 mm. thick, and dark reddish-brown in color; the pyramids are congested. The calyces, pelvis, and ureter are normal. The left kidney weighs 170 grammes, and presents appearances similar to its fellow. The adrenals are normal. The bladder contains a small quantity of clear urine; its mucous membrane and wall are normal. The stomach is normal in size and position. It contains some turbid brownish fluid. The mucous membrane is congested and somewhat swollen. The duodenum and the remainder of the intestine are normal. The brain and spinal cord were not examined.

The anatomical diagnosis was as follows: acute hemorrhagic peritonitis; chronic adhesive pericarditis, with extensive calcification; chronic bilateral adhesive pleuritis; chronic perihepatitis (pericarditic pseudocirrhosis of the liver of Pick); slight congestion of the liver, spleen, and kidneys.

Microscopic examination of the thickened pericardium revealed marked overgrowth of fibrous connective tissue with relatively few cells, and some extension of the newly-formed connective tissue into the interstices of the heart muscle. Careful examinations of sections from several regions revealed no evidences of tuberculosis. Examination of the thickened pleuræ revealed only fibroid thickening; no evidences of tuberculosis. Examination of the liver revealed chronic fibroid thickening of the capsule and some slight extension of connective tissue trabeculæ a short distance into the substance of the liver. In addition, evidences of acute inflammation were apparent, the round-cell infiltration being rather abundant. No evidence of tuberculosis could be detected. The liver itself revealed moderate distention of the central veins of the lobules and of the adjoining capillaries, with a moderate atrophy of the intervening liver cells. The cells of the periphery of the lobules showed slight fatty infiltration. The interlobular connective tissue possibly was slightly in excess of the normal, and contained a slight amount of round-cell infiltration. The spleen revealed the usual evidences of slight congestion and slight thickening of the trabeculæ.

The occurrence of marked ascites, with little or no œdema of the extremities in cases of failing cardiac compensation, though not very common, is by no means unknown. It was mentioned by Liebermeister in 1864, by Oppolzer in 1866, and it is observed by most clinicians of experience at the present time. The difficulties attending the diagnosis in these cases, however, are sometimes very great. The question whether an enlarged liver (in advanced cases a small liver) with ascites is to be referred to a primary or a secondary liver disease, if it presents itself to the mind of the clinician at all, may be difficult of solution. Light is shed on the case by the detecting of trustworthy evidence of cardiac valvular disease—in which case, especially if the patient be an adult, one has to determine whether the liver condition and the ascites are due to the heart disease or to an associated alcoholic cirrhosis of the liver. The diagnosis is even more difficult in cases of obliterative pericarditis, which appears to give rise to disproportionate ascites more frequently than does valvular disease of the heart. In these cases the pericarditis being usually latent and more or less insusceptible of diagnosis, the ascites is generally referred to a cirrhosis of the liver, rarely to tuberculous peritonitis, etc.

The cause of disproportionate ascites in cases of obliterative pericarditis has occasioned much discussion. Thus Weiss attributed it to changes in the bloodvessels of the peritoneum, the result of chronic peritonitis. He believed that in consequence of the pericardial obliteration general venous congestion occurs; that in consequence of the inflammation of the peritoneum, the peritoneum becomes a punctum minimæ resistentiæ; that transudation of serum occurs more readily from the altered vessels of the peritoneum than from other vessels of the body;

and that in consequence of the development of the ascites venous congestion in other parts of the body is relieved, and the development of œdema more or less prevented.

Mott believed that owing to the adherent pericardium interfering with the coronary circulation, the heart, instead of undergoing compensatory hypertrophy, as it sometimes does in the condition under discussion, slowly undergoes degeneration, and that this is doubtless hastened by the immense loss of fluid. He stated that, moreover, the thick adherent pericardium would interfere considerably with the elasticity of the walls of the heart and greatly modify its action as a suction pump during diastole, and that the circulation that this would affect most would be the portal, because here the blood has to pass through two sets of capillaries and against gravity. This is a view that is scarcely tenable, since, were it true, we should expect predominating ascites in all cases of failing cardiac compensation.

Weinberg attributed the ascites, with little or no œdema of the legs, to angulation of the inferior vena cava or the hepatic vein due to a right-sided pleural effusion. He assumed that in consequence of the angulation of the veins congestion and other changes in the liver, with consecutive ascites, occurred; and he endeavored to support his opinions by citing the results of some experiments of Rosenbach's that appeared to show that such right-sided pleural effusion may cause angulation of the vena cava and congestion of the portal area. However, as pointed out by Pick, it is difficult to conceive of isolated compression of the hepatic vein, and compression or angulation of the inferior vena cava results in œdema of the legs rather than in ascites. Furthermore, in many of the cases there is no right-sided pleural effusion.

Rosenbach, while scarcely venturing to express a positive opinion on the subject, states that the ascites cannot be the result of simple insufficiency of the heart muscle, and he attributes considerable importance to the fibrous alterations in the liver capsule. He says, further, that the ascites must be the result of the operation of special conditions which, though associated with the heart affection, are not the direct result of diminution of the power of the heart; and that probably we have to do with an extension of the inflammatory process from the pericardium along the large veins to the serous covering of the liver, or vice versa, with the final development of connective tissue hyperplasia.

Pick, criticising adversely the opinions of Weiss, Mott, and Weinberg, points out that each of these writers in attempting to explain the occurrence of the ascites entirely overlooked that organ which, from the clinical course of the disease, one would be inclined to think most at fault—the liver. He states that in these cases the liver is described as rounded, increased in consistency, hemorrhagic, enlarged, markedly irregular, granular, very firm, cirrhotic—in short, the appearances of the different stages of cyanotic induration, atrophic nutmeg liver, or the cardiac cirrhosis of the French. And inasmuch as other writers have observed prevailing ascites rather than œdema of the extremities in certain forms of heart disease, with similar secondary changes in the liver, he prefers to attribute the ascites in these cases of obliterative pericarditis to the alterations in the liver rather than to assume certain hypotheses. He says, further, that the question why in these cases ascites rather than generalized œdema occurs is intimately bound up with the question why in certain cases of congestion of the liver, in failing compensation of the heart in valvular disease, connective tissue proliferation occurs, whereas in other cases it does not occur; and that concerning this we know little definitely. For the time being he was content to point out the diagnostic features of the symptom complex in question. He believes that the associated peritoneal alterations are more or less accidental—that the thickening of the peritoneum results from the long-continued congestion and the persistence of the ascites, and that the thickening and the adhesions are in part due also to infection the result of repeated tappings.

Harris states: "There are two lesions, either of which may possibly occur as complications to the mediastinal and pericardial affection, and which could explain the ascites in such cases. In the first place, it is conceivable that chronic venous congestion of the liver produced by the cardiac dilatation may in some cases set up a chronic inflammation of the liver, a secondary periportal cirrhosis, which like the ordinary alcoholic cirrhosis would obstruct the portal circulation and produce ascites. We have seen that an increase of fibrous tissue in the liver is found in some of the cases of mediastino-pericarditis; but such a change does not appear to be the rule, and in the cases where it has been found it is questionable whether it has been sufficiently pronounced to produce portal obstruction and ascites therefrom. Furthermore, in one of my cases (Case II.) where the abdominal dropsy was such a prominent feature of the illness, the ascites was certainly not due to a cirrhotic liver, no marked periportal cirrhosis being found on microscopic examination. The second explanation of the occurrence of the ascites in the cases under consideration is that a chronic peritonitis ensues, and to it the ascites is due. Such chronic peritonitis in some instances may possibly be an independent affection, and one not directly connected with the mediastinal or pericardial lesion; in other cases it is conceivable that the chronic venous congestion due to the intrathoracic affection sets up the chronic peritonitis. Such a chronic peritoneal change was found in my second case, and was clearly sufficient to account for the ascites. That it is the explanation of the ascites in all such cases, of course, we do not say. It is quite possible that other complications may, in some cases, occur which account for the development of ascites as a prominent feature of the case." After further discussion of the question, he says: "I am inclined, therefore, to think that the ascites which sometimes occurs in cases of mediastino-pericarditis, and which is unaccompanied by extreme anasarca of the lower extremities, is due, in some instances, at all events, to the onset of a peculiar form of chronic peritonitis. This chronic peritonitis may be possibly due to venous engorgement of the peritoneum consequent upon the dilatation of the heart or venous obstruction in the mediastinum, or it may occur so early in the case as to be probably independent of such venous stasis. In my second case the ascites was a prominent symptom from a very early period of the illness, and probably was independent of venous engorgement, which only appeared as a marked feature some time after the development of the ascites."

Heidemann, supporting the opinion of Weiss, states that in these cases we have to deal with a chronic inflammation of divers serous membranes; that the congestion, the result of the degeneration of the muscle of the heart, leads to ascites because the vessels of the peritoneum, on account of the chronic inflammation, constitute a locus minoris resistentiæ; that the associated cirrhotic processes in the liver observed in these cases result from an extension of the inflammatory irritant from the liver capsule and from the chronic congestion of the organ; and that in consequence of proliferation and shrinkage of the connective tissue on and in the liver the congestion and exudation into the peritoneal cavity are increased.

Eisenmenger, a pupil of Paltauf, states that it is quite true that in consequence of obliterative pericarditis marked ascites, with little or no œdema of the legs, occurs comparatively frequently, and that if the pericarditis is latent, the symptom complex bears a certain resemblance to cirrhosis of the liver. He states further that this symptom complex is not due, as is asserted by Pick, to connective tissue proliferation the result of disturbances of circulation in the liver, but that in different cases it is due to different causes. He emphasizes the etiological importance of distortions, compression, and angulations of the inferior vena cava, produced by a concomitant exudate or pleuropericardial thickenings; concomitant peritonitis in the transverse fissure of the liver; and, because the symptom complex is observed especially in young persons, the condition of the capillaries and small vessels in healthy young persons, in consequence of which œdematous transudations occur readily. Inasmuch, then, as the disease is not an anatomical entity, he disapproves of the designation "pericarditic pseudocirrhosis of the liver," suggested by Pick.

Without detailing the opinions of other writers, it suffices to state that the opinions of Mott and Weinberg are scarcely worthy of serious consideration. In addition to the opinion of Eisenmenger, which is not supported by the report of any illustrative cases, there remain three opinions held by as many different groups of writers: the first, represented by Pick, Bozzolo, Nachod, Galvagni, Cabot, and others, attributes the ascites to changes in the liver the result of long-standing congestion; the second, represented by Weiss, Heidemann, Schupfer, Werbatus, and others, attributes the ascites primarily to the chronic peritonitis; and the third, represented by Harris, Osler, Patella, Siegert, and others, attributes the ascites to the combined action of both of these factors.

In view of these differences of opinion, it may not be unprofitable, from a study of the cases reported in the literature, to ascertain the more likely cause of the predominating ascites, and in general the nature of the disease in question. From the subjoined table I have omitted all cases in which the ascites was merely a part of generalized dropsy and effusions into the several serous cavities of the body; cases, of which Wagner's and Türk's serve as examples, in which the lesions were a disseminated tuberculosis of the organs, as well as of the serous membranes; and, for instance, a second case reported by Diemer, in which obliterative pericarditis and atrophic cirrhosis of the liver occurred, but in which there was no note of ascites. I have been obliged reluctantly to omit also a number of cases reported by Italians —Galvagni, Bozzolo, Patella, and Caporali. Their reports were unavailable in the original, and the abstracts at my disposal were not sufficiently complete to enable me to incorporate them in the table.

Reporter.	Sex and age.	Duration, years.	Ascites.	Œdema.	Pericardium.	Right pleura.	Left pleura.	Peritoneum.	Liver.	Spleen.	Remarks.
Van Deen, 1840	M. 40	2	Constant.	Subsequent to ascites.	Markedly thickened and firmly adherent to the heart.	Adherent throughout	Adherent throughout	Thickened throughout; many adhesions.	Chronic perihepatitis; liver otherwise normal.	Chronic perisplenitis; spleen otherwise normal.	The peritoneum of the under surface of the diaphragm and of the upper portion of the abdomen was least affected; pericarditis believed to be the primary affection, the pleuræ and the peritoneum being affected secondarily, the peritoneum through spreading of the inflammation through the diaphragm.
Frerichs, 1861	F. 38	2	Constant.	Constant, but moderate.	Firmly adherent throughout.	Adherent throughout	Adherent throughout	Cloudy, injected, and much thickened in many places.	Marked chronic perihepatitis; cirrhosis; liver one-third smaller than normal.	Marked chronic perisplenitis; spleen enlarged.	It is believed that the liver induration was caused by a peritonitis, the symptoms of which preceded by two years symptoms referable to the liver.
Feierabend, 1866	M. 50	4	Constant.	No.	Adher'nt and markedly calcified, especially the anterior surface; the apex free.	....	....	....	Marked cirrhosis.	Enlarged.	Pericarditis 23 years previously; four tappings for relief of the ascites; the clinical diagnosis was cirrhosis of the liver with secondary gastro-intestinal catarrh.
Hambursin, 1869	M. 50	4	Constant.	Only shortly before death.	Chronic adhesions to the right of the base of the heart and to diaphragm; also fluid.	Chronic adhesions.	Normal.	Chronic adhesions and thickening.	Marked chronic perihepatitis; liver small, nutmeg, and cirrhotic.	Normal.	The disease is presumed to have begun in the liver capsule and to have implicated the pericardium subsequently.
Weiss, 1876	F. 51	1½	Constant.	Constant; moderate.	Thickened and adherent throughout.	Obliterated	Obliterated	Thickened & opaque; many adhesions.	Chronic perihepatitis; liver large and firm.	Chronic perisplenitis.	Presumed to have followed rheumatism; the anatomical diagnosis was total concretion of the abdominal and thoracic organs.
Weiss, 1876	F. 14	2	Constant	Only shortly before death.	Obliterated.	Fluid.	Obliterated	Thickened & opaque; many adhesions.	Chronic perihepatitis; liver firm.	Chronic perisplenitis.	Followed typhoid fever; many tappings for relief of the ascites.
Vierordt, 1884	F. 20	6	Constant.	Slight late in the course of the disease.	Obliterated.	Chronic adhesions.	Normal.	Thickened & opaque; many adhesions	Chronic perihepatitis; cirrhosis.	Chronic perisplenitis; spleen enlarged.	Presumed to have begun in the liver capsule; standstill for 1 year; 34 tappings (460 litres of fluid) for relief of the ascites.

Vierordt, 1884	F. 35	6	Constant.	Slight and transient.	Obliterated; marked calcification.	Obliterat'd; thickened.	Obliterat'd; thickened.	Thickened & opaque; many adhesions.	Marked chronic perihepatitis (zuckergussleber); liver nutmeg.	Marked chronic perisplenitis (zuckergussmilz); spleen enlarged.	Presumed to have started in the pericardium; standstill for several years; several tappings for relief of the ascites; it is suggested that the peritonitis and the chronic inflammatory changes in the pericardium stand in some relationship to each other.
Curschmann, 1884	F. 54	6½	Constant.	No.	Obliterated; thickened.	Obliterat'd; thickened.	Acute serofibrinous inflammation.	Thickening, induration, opacity, and adhesions of the peritoneum of the upper half of the abdomen.	Diffuse chronic hyperplastic perihepatitis; compression and reduction in size (⅓) of the otherwise unaltered liver.	Marked chronic perisplenitis; spleen double the normal size.	Many tappings for relief of the ascites; standstill for a year and a half; recent tuberculous peritonitis at the necropsy.
Tissier, 1885	F. 51	10	Constant.	Yes.	Obliterated; calcification at left edge anteriorly & posteriorly; apex free.	Fluid.	Fluid.	Thickening, opacity and adhesions.	Marked chronic perihepatitis (zuckergussleber); liver en-enlarged and nutmeg.	Chronic perisplenitis.	Several punctures for relief of the ascites.
Hirschler, 1886	F. 48	...	Constant.	Slight.	Obliterated.	Fluid.					
Weinberg, 1887	M. 19	⅓	Constant, though preceded by symptoms of acute pleuritis.	Subsequent to the ascites.	Thickened and obliterated.	Fluid.	Fluid.	Chronic peritonitis.	Chronic perihepatitis; liver nutmeg.	Enlarged.	Two tappings for relief of the ascites; process tuberculous, the result of primary infection of the pericardium by a tuberculous bronchial lymph gland.
Weinberg, 1887	F. 18	⅔	Constant.	Late in the course of the disease.	Thickened and obliterated.	Obliterated	Obliterated	Thickening, opacity and adhesions.	Chronic perihepatitis (not so marked on diaphragmatic surface as elsewhere); liver congested; weight 2050 grammes.	Normal.	Fifteen tappings for relief of the ascites; process tuberculous, the result of primary infection of the pericardium by tuberculous bronchial lymph gland.
Mott, 1887	M. 17	2½	Constant.	Only shortly before death.	Thickened and obliterated.	Thickened and obliterated.	Thickened and obliterated.	Inflammatory lymph	Nutmeg; weight 54 ounces.	Normal.	Several tappings for relief of the ascites; process began as a pleuropericarditis.
Mott, 1887	M. 23	1½	Constant.	Subsequent to and less than the ascites.	Thickened and obliterated.	Recent serofibrinous exudate.	Thickened and obliterated.	....	Enlarged; weight 5 pounds.	Congested; weight 18 oz.	Process began as a pleuropericarditis.

Reporter.	Sex and age.	Duration, years	Ascites.	Œdema.	Pericardium.	Right pleura.	Left pleura.	Peritoneum.	Liver.	Spleen.	Remarks.
Variot, 1888	M. 51	...	Constant.	....	Obliterated; marked calcification of anterior surface of right ventricle, left auricle, right edge, and posterior surface.	Chronic adhesions and fluid.	Chronic adhesions.	Chronic adhesions.	Marked chronic perihepatitis (zuckergussleber); liver enlarged.	Somewhat enlarged.	Clinical diagnosis: mitral insufficiency and right-sided pleural effusion.
Riedel, 1892	M. 19	15	Constant.	Only shortly before death.	Thickened and obliterated.	Chronic adhesions, thickening and fluid.	Chronic adhesions, thick'ning, and fluid.	Entire peritoneum markedly thickened and adherent.	Marked chronic perihepatitis (zuckergussleber) with extension of connective tissue into the liver substance.	Chronic perisplenitis: spleen enlarged.	Heart, lungs, liver, and spleen—zuckerguss; several tappings for relief of the ascites. Process presumed to have begun in the different serous membranes at the same time, although it is admitted that the disease may have begun in the pericardium as a complication of pertussis, and subsequently have implicated the other serous membranes by contiguity of structure. Standstill for eight years.
Cantu, 1894	F. 41	...	Constant.	....	Thickened and obliterated.	Fluid.	Fluid.	....	Nutmeg.	Congested.	
Rumpf, 1895	F. 40	16	Constant.	Of left leg.	Thickened and obliterated.	Chronic adhesions and fluid.	Chronic adhesions and thickening.	Thickening, opacity and adhesions.	Diffuse chronic hyperplastic perinephritis (zuckergussleber); compression of the otherwise unaltered liver.	Chronic perisplenitis; spleen moderately enlarged.	301 tappings for relief of the ascites; process presumed to have begun in the pericardium; acute pericarditis at the age of 33 years.
Henoch, 1895	M. 5	14 days (?)	Constant.	Constant.	Thickened and obliterated.	Thickened and adherent.	Thickened and adherent.	....	Chronic perihepatitis; liver enlarged and cirrhotic.	Enlarged.	Presumed to be syphilitic.
Harris, 1895	M. 8	3½	Constant.	Slight.	Thickened and obliterated.	Thickened and obliterated.	Thickened and obliterated.	Thickening, opacity and adhesions.	Marked chronic perihepatitis; liver nutmeg; little or no cirrhosis.	Marked chronic perisplenitis; spleen congested.	Many tappings for relief of the ascites; old tuberculous focus in the apex of the right lung; tuberculous arthritis of the right elbow.
Broadbent, 1895	F. 16	½	Constant.	Present, but much less than the ascites.	Thickened and obliterated.	....	....	Recent miliary tuberculosis.	Enlarged and nutmeg.	....	Tuberculous focus in the apex of the left lung; striking feature of the case "the constant accumulation of fluid in the peritoneal cavity."

Pick, * 1896	M. 47	4	Constant.	Slight.	Thickened and obliterated.	Fluid.	Chronic adhesions.	General fibrous peritonitis.	Chronic perihepatitis; liver enlarged and nutmeg.	Chronic perisplenitis; spleen congested.	Many tappings for relief of the ascites.
Pick, 1896	M. 26	8	Constant.	Late in the course of the disease.	Thickened and obliterated; extensive calcification.	Fluid.	Fluid.	....	Enlarged and cirrhotic.	Enlarged five times the normal size.	Many tappings for relief of the ascites.
Pick, 1896	M. 24	2	Constant.	Subsequent to the ascites.	Obliterated; extensive calcification.	Chronic adhesions.	Chronic adhesions.	Thickening, opacity and adhesions.	Perihepatitis and cirrhosis.	Perisplenitis.	Process tuberculous; correct diagnosis during life.
Osler, 1896	F. 14	3½	Constant.	General at first; disappeared; reappeared; but always slight.	Thickened and partly obliterated.	Chronic adhesions.	....	Thickening, opacity and adhesions.	Marked chronic perihepatitis and cirrhosis.	Perisplenitis; spleen enlarged and congested.	Correct diagnosis during life; extreme cyanosis of the hands and feet, and subcutaneous nodules about the knuckles, wrists, and elbows; 121 tappings for relief of the ascites.
Dickinson, 1896	F. 17	5	Constant.	Much less than the ascites.	Thickened and obliterated.	Chronic adhesions.	Obliterated	....	Enlarged (64 oz.) and nutmeg; capsule thickened.	....	26 tappings for relief of the ascites.
Heidemann, 1897	F. 15	1	Constant.	Subsequent to the ascites.	Obliterated.	Chronic adhesions and fluid.	Serofibrinous exudate.	Fibrous peritonitis, especially of Douglas' cul-de-sac.	Small nutmeg and cirrhotic.	Somewhat enlarged.	Pericarditis one year previously; 23 tappings for relief of the ascites.
Schupler, 1897	M. 44	4¼	Constant.	Slight at beginning; disappear'd and recurred shortly before death.	Obliterated.	Thickened and obliterated.	Thickened and obliterated.	Chronic fibrous hyperplastic peritonitis.	Marked chronic perihepatitis with extension of the connective tissue into the liver substance.	Perisplenitis.	Presumed to have begun at the same time in the different serous membranes; standstill for one year.
Cabot, 1898	M. 18	6	Constant.	Slight at intervals.	Obliterated.	Chronic adhesions and fluid.	Chronic adhesions and fluid.	....	Enlarged and nutmeg.	....	Several tappings for relief of the ascites.
Siegert, 1898	M. 19	9	Constant.	General at the beginning; disappeared, and recurred for short periods, & again before death.	Thickened and obliterated.	Chronic adhesions.	Chronic adhesions.	Thickening, opacity and adhesions.	Chronic perihepatitis with extension of connective tissue into the liver substance; liver congested.	Perisplenitis; spleen slightly enlarged.	Presumed to have begun in the pericardium; standstill for two and a half years; more than 100 tappings for relief of ascites.
Werbatus, 1898	M. 42	6	Constant.	Subsequent to the ascites.	Thickened and obliterated.	Thickened and obliterated.	Thickened and obliterated.	Thickening, opacity and adhesions.	Chronic perihepatitis; atrophic nutmeg liver and cirrhosis.	Chronic perisplenitis and congestion.	

Reporter.	Sex and age.	Duration, years.	Ascites.	Œdema.	Pericardium.	Right pleura.	Left pleura.	Peritoneum.	Liver.	Spleen.	Remarks.
Nachod, *1898	M. 6	1	Constant.	Slight at beginning; disappear'd and reappeared shortly before death.	Thickened and obliterated.	Extensive adhesions.	Extensive adhesions.	Chronic adhesions.	Liver firmer than normal; many caseous nodules in the capsule.	Enlarged; capsule thickened and contained many caseous nodules.	Nine months prior to death, with the idea that the ascites was due to tuberculous peritonitis, cœliotomy was performed, but the peritoneum was found entirely normal; the pericarditis evidently was primary; some bronchial glands were tuberculous.
Taylor, 1898	F. 42	1½	Constant.	Subsequent to the ascites.	Thickened and obliterated.	Thickened and obliterated.	Thickened and obliterated.	Univers'lly thick and opaque.	Perihepatitis and compression of the liver.	Perisplenitis; spleen hard and small.	
Ewart, 1899	F. 49	4	Constant.	Slight.	Obliterated and calcified.	....	....	Adhesions and thickening.	Enlarged and congested.	Perisplenitis.	Operated upon for the cure of a supposed cirrhosis of the liver; previously to the operation fifty-two tappings for the relief of the ascites.
Diomer, 1899	M. 38	1½	Constant.	Present at first; disappeared, and reappeared.	Obliterated; extensive calcification, wide calcareous circlet about both ventricles; apex free.	Thickening and chronic adhesions.	Normal.	Diffuse thickening and adhesions.	Chronic fibrous perihepatitis (zuckergussleber); diffuse syphilitic cirrhosis.	Chronic perisplenitis (zuckergussmilz); chronic congestion.	Many tappings for relief of the ascites.
Schmaltz and Weber, 1899	F. 42	5	Constant.	Slight subsequent to the ascites.	Obliterated by recent adhesions.	Slight adhesions.	Adhesions.	Chronic adhesions.	Marked chronic perihepatitis (zuckergussleber); no connective tissue increase or marked congestion.	Marked chronic perisplenitis (zuckergussmilz); congestion.	Many tappings for relief of the ascites; standstill for two years.
Herrick, 1902	M. 20	4	Constant.	Present at first; disappeared and recurred.	Obliterated; extensive calcification of left half anteriorly & posteriorly, and of diaphragmatic surface.	Obliterated and thickened; small tuberculous sac.	Obliterated and thickened.	Smooth except for the perihepatitis and perisplenitis.	Chronic perihepatitis; liver firm; weight 2235 grammes; microscopic examination showed "cardiac cirrhosis."	Perisplenitis; spleen greatly enlarged.	Process tuberculous; several tappings for relief of the ascites and the pleural effusion; standstill for one year.
Kelly, 1902	M. 31	7	Constant.	Slight and transitory.	Obliterated; extensive calcification of both ventricles.	Thickened and obliterated.	Thickened and obliterated.	Recent hemorrhagic peritonitis	Chronic perihepatitis; congestion.	Congestion.	Several tappings for relief of the ascites.

Of the 39 cases in the table, 21 occurred in males, and 18 in females. The ages of the patients were as follows: 3 were under ten years of age; 13 were between eleven and twenty years; 3 were between twenty-one and thirty years; 5 were between thirty-one and forty years; 11 were between forty-one and fifty years; and 4 were between fifty-one and fifty-four years. This summary well illustrates a fact commented upon by a number of writers, namely, the prevalence of the disease among young subjects. Presumably, it indicates also that should the patient survive the twentieth year he or she is likely to live until the fourth decade of life. In 3 of the cases the duration of the disease was not ascertainable. In 26 cases the duration was two years or more; in 19 cases it was four years or more; in 1 case it was ten years; in 1 case it was fifteen years; and in 1 case it was sixteen years. In 1 case (Henoch's) the duration is put questionably at fourteen days. The striking clinical feature of all of the cases was the constant and predominating ascites, an ascites that necessitated many tappings and continuously recurred—whence the diagnosis in many cases of cirrhosis of the liver, in other cases of peritoneal tuberculosis. In most of the cases œdema of the legs was absent until shortly before death. In a few cases œdema was slight throughout the course of the disease, and in several of these it increased moderately or considerably shortly before death. In other cases it was present early in the course of the disease, but it soon disappeared, although the ascites persisted, and it did not recur until shortly before death; or it reappeared and disappeared irregularly throughout the course of the disease.

With regard to the anatomical lesions—in one case (Schmaltz and Weber's) the pericardium was obliterated by recent adhesions. In the remaining 38 cases the pericardium was thickened and obliterated by chronic adhesions—in almost all of the cases entirely obliterated, in one or two cases only partly obliterated. In 10 of the cases there was, in addition, more or less extensive calcification of the pericardium —the case herewith reported being the most marked example of this condition that I have encountered.

There is no statement with regard to the condition of the right pleura in 3 cases. In 1 case the right pleura contained recent serofibrinous exudate, in 6 cases it contained fluid, in 12 cases there were more or less extensive chronic adhesions, and in the remaining 17 cases the pleura was entirely obliterated and generally thickened. There is no statement with regard to the condition of the left pleura in 5 cases. In 3 cases the left pleura was normal, in 2 cases it contained recent serofibrinous exudate, in 4 cases it contained fluid, in 10 cases there were more or less extensive chronic adhesions, and in the remaining 15 cases the pleura was entirely obliterated and generally thickened. In most of the cases in which there were chronic adhesions fluid also was present, and in most of the cases in which there were thickening and obliteration of the pleura and pericardial sacs there were also external adhesions binding the pericardium to the chest wall and external surface of the pleura and the adjacent tissues of the mediastinum. The summary well illustrates the more marked involvement of the right pleura as contrasted with the left pleura—a subject that has been much commented upon recently. There is no statement with regard to the condition of the general peritoneum in 8 cases. In 1 case there was recent hemorrhagic peritonitis, in 1 case recent miliary tuberculosis, and in 1 case inflammatory lymph, and in the remaining 28 cases there were more or less extensive chronic adhesions.

There is no statement with regard to the condition of the liver in 1 case. In 28 cases chronic perihepatitis was noted, and in 31 cases there was either chronic perihepatitis or chronic peritonitis or both. The liver itself was reported to be nutmeg in appearance in 14 cases, cirrhotic in 8 cases, large and firm in 5 cases, normal aside from compression in 4 cases, nutmeg and cirrhotic in 4 cases, and in 3 cases there appeared to be only extension of connective tissue from the thickened capsule into the substance of the organ. Perisplenitis was noted in 23 cases. There was no statement with regard to the condition of the spleen itself in 12 cases. In 23 cases it was congested, in 1 case it was small and hard, and in 3 cases it was normal.

From this tabulation and summary two facts stand out prominently: 1. That although all the cases presented ascites as the striking clinical feature and revealed obliterative pericarditis at the necropsy, yet that organ that we might presume to be most at fault—the liver—presented a variety of lesions in the different cases; and 2, that in all the cases more than one serous membrane, in some of the cases all the serous membranes were diseased. The feature possessed by the cases in common, therefore, is widespread disease of the serous membranes, and the most common lesion is chronic hyperplastic serositis.

In some cases there is a clear history of the repeated occurrence of inflammation of one or more serous membranes; in other cases the disease was latent and entirely unsuspected during life. In some cases the history points definitely to a certain serous membrane as the point of departure of the disease, to the membrane whence the inflammatory process spread to the other membranes. In this connection, however, we should bear in mind that the serous membrane disease that attracted attention in some of these cases began acutely, and that such disease of a serous membrane may just as well be the consequence as the cause of a latent inflammation of some other serous membrane. Thus it is that in some of the cases with multiple involvement of the serous membranes, different writers interpret the necropsy findings differently. Some attribute the symptom complex to a primary pericarditis with consecutive pleuritis and peritonitis; others hold the opposite view, and look upon the peritonitis as the primary event, and attribute the pleuritis and pericarditis to extension of the inflammation through the diaphragm from a perihepatitis; others hold that the perihepatitis is the primary disease and that from this by continuity and contiguity of structure the peritonitis and the pericarditis arise; and, finally, still others hold that all of the serous membranes become infected with the same noxious agent at the same time.

From this point of view Picchini distinguishes four groups of cases of multiple serositis or polyserositis: 1. Cases in which by means of careful inquiry the point of origin of the disease, that is, the serous membrane first affected, as well as the order of the involvement of the other serous membranes, may be determined. 2. Cases in which on inquiry the point of origin of the disease may be determined, but in which the first examination of the patient reveals that several serous membranes are already affected, although they manifest no clinical symptoms. 3. Cases in which the history reveals that from the beginning several serous membranes have been affected. 4. Cases in which from the history it cannot be determined whether the disease began with inflammation of one or more serous membranes. Of the 50 cases observed by Picchini, 37 belonged to the first group; 11 to the second; 1 to the third, and 1 to the fourth group. Of 110 collected by Picchini (50 observed by himself and 60 collected from the literature) the peritoneum was involved in all, although this was recognized clinically in only 13 of the cases. This peritonitis was not only the most frequent, but also the most important lesion; it dominated the situation. Pleuritis was present in 109 cases, in 73 of which it was bilateral. The pericardium was least frequently involved—only in 9 of 50 cases. This fact is deemed evidence of great resistance on the part of the pericardium.

De Renzi states that in multiple serositis the peritoneum is usually involved first; then the right pleura, and then the pericardium. If, however, the right pleura should be involved first, the disease then extends to the peritoneum, thence to the left pleura and the pericardium. In some cases, however, this mode of progression of the disease is not preserved, as sometimes a pericarditis that usually is not to be diagnosticated develops first. De Renzi states further that the disease is characterized by the fact that it remains localized to the serous membranes; that it does not implicate the intra-abdominal and intrathoracic organs; that it pursues a remarkably slow and insidious course; that it gives rise to the exudation of large quantities of serofibrinous fluid; and that the fluid portion of the exudate tends to become absorbed, in consequence of which fibrous adhesions with complete obliteration of the serous cavities result.

Presuming, then, that the inflammation may begin in any serous membrane, it is evident that the clinical manifestations may vary, depending upon the serous membrane first affected. This is commented upon by a number of writers, and it is discussed at length by Siegert, who points out that although the chronic perihepatitis and the resultant ascites dominate the clinical picture, the clinical picture nevertheless assumes quite different aspects depending upon whether the disease begins in one or more serous membranes. Thus, if the liver capsule is first affected ascites occurs as an inflammatory exudate. If after the lapse of some time the perihepatitis leads to compression of the liver and the portal radicles, another factor contributing to congestion and ascites is added. Thus it is that ascites is the prominent symptom in those cases of multiple serositis in which the disease begins in the peritoneum. Later in the course of the disease the marked increase in the intra-abdominal pressure occasioned by the large amount of the ascites, as well as the spread of the inflammatory process to the pleura and the pericardium, may lead to congestion in the area of collection of the inferior vena cava, so that to the ascites œdema of the legs is added.

That multiple serositis may begin in one or the other pleura must be conceded, and that such is really the case is rendered likely by the history of early and apparently primary pleuritis in several of the cases tabulated. Especial interest, however, attaches to primary involvement of the pericardium. In the reports of some of the cases tabulated there appears a note that acute pericarditis occurred some years prior to the development of the symptoms that interest us at present, and in other of the cases the nature and the extent of pericardial lesions warrant the assumption that these were the primary lesions—for instance, the cases reported by Feierabend, Vierordt, Weinberg, Mott, Riedel (?), Rumpf, Pick, Osler, Heidemann, Siegert, Nachod, Ewart, and my own case. When the pericardium is primarily affected certain features in the clinical course of the disease may occur that possess especial interest and diagnostic importance. Whereas in those cases in which the peritonitis or perihepatitis is the primary event, ascites occurs early and is succeeded by œdema only late in the course of the disease, in the cases beginning with pericarditis œdema of the legs may occur early—the consequence of early and transitory insufficiency of the myocardium. In cases in which this occurs the œdema usually soon disappears and is succeeded by ascites, which assumes and maintains the ascendency—for instance, cases reported by Vierordt, Broadbent, Rumpf, Osler, Dickinson, Schupfer, Siegert, Nachod, and Diemer. However, as pointed out by Siegert, the absence of œdema of the legs in the presence of ascites early in the course of the disease is no evidence of primary implication of the peritoneum rather than of the pericardium, since it is well known that chronic obliterative pericarditis frequently runs a course entirely devoid of symptoms.

The cause of this obliterative pericarditis, as well as the cause of the multiple serositis in general, has occasioned considerable discussion. Certain of the infective diseases, such as typhoid fever, pertussis, malaria, syphilis, and rheumatism, are given as etiological factors in a number of the cases tabulated. In a number of the cases the lesions are of extremely obscure or unknown etiology, whereas, in other cases the provoking agent was certainly the tubercle bacillus. The fact that possesses considerable interest in this connection is that during recent years medical writers have become more and more inclined to look upon many cases of chronic obliterative pericarditis (as well as disease of other serous membranes) as tuberculous in nature, and this even in the absence of clear evidence of tuberculosis. This has been insisted upon for a number of years by the German and the Italian writers, and it is emphasized by Riesman and by Wells in their recent communications. According to a number of writers the disease is always tuberculous in nature; other writers, however, are disposed to admit the occasional operation of other etiological factors, inasmuch as it has not been possible to demonstrate the tuberculous nature of all cases by either macroscopic, microscopic, or bacteriologic examinations. The important fact to bear in mind is that many cases apparently non-tuberculous are in reality due to the tubercle bacillus, and that in some cases characteristic tuberculous lesions may be detected by microscopic examination of the tissues, and that the tubercle bacillus may be recovered from some of the lesions by appropriate bacteriologic and inoculation procedures. Presumably, also, the patient would react to tuberculin injections. On the other hand, that a number of cases are not tuberculous must be conceded. In my own case careful microscopic examination of the tissues failed to disclose any tuberculous lesions. I would suggest that in my own case, as well as in other cases, antecedent rheumatism possesses considerable etiological importance. In some cases chronic nephritis may be the etiological factor; whereas in other cases the disease seems to develop from cholelithiasis with cholecystitis and pericholecystitis (localized peritonitis).

An important and interesting fact especially insisted upon by the Italian writers is that not every case of general tuberculosis that implicates the serous membranes and gives rise to chronic fibrous lesions belongs to the group of cases under discussion. Picchini states that the cases under discussion differ from the ordinary cases of serous membrane tuberculosis in that in their etiology heredity appears to play no rôle; that the disease does not appear to affect especially persons of phthisical or tuberculous habitus; that the inflammations of the serous membranes set in insidiously, much less acutely than do the ordinary cases of tuberculous inflammation; that they may remain latent for a long time; and that the lesions remain localized to the serous investment of the organs and are unassociated with tuberculous lesions of the lungs, as is so common in the ordinary cases. A study of the cases reported in the literature bears out the reasonableness of this view.

A marked susceptibility of the serous membranes and a certain immunity of the organs to the cause of the disease—presumably the tubercle bacillus in many of the cases—appear to be striking features of the cases. Nevertheless, there seem to be border-line cases and cases representing varying degrees of infection with the tubercle bacillus: 1. Cases in which the lesions are confined strictly to the serous membranes. 2. Cases in which the lesions involve, especially the serous membranes, but in which one or several tuberculous foci may be found in the body—for instance, the cases reported by Weinberg, Harris, Broadbent, Pick, and Nachod. 3. Cases in which the serous membrane disease represents but a part of a general tuberculous infection (not considered in this communication). That this is probably true is borne out by the fact that in one or two of the cases the fatal termination was brought about by an acute disseminated miliary tuberculosis. Cantu accounts for the peculiarities of the disease as contrasted with the ordinary cases of tuberculosis of the serous membranes by assuming that it is due to a special form of the tubercle bacillus. On account of the length and arrangement of the tubercle bacilli in the newly formed connective tissue in his case, he assumed that the bacillus operative was allied to that form peculiar to birds.

Some discussion has been occasioned by certain writers—Pick, Schupfer, Werbatus, Heidemann, and others—including the group of cases under consideration, the cases of "zuckergussleber," reported by Curschmann, Rumpf, and Hübler. Reference to the table will show that the cases of Curschmann and of Rumpf are in reality cases of multiple serositis, that in them the lesions of the serous membranes were widespread. In Hübler's case, as well as in a number of cases reported by Hale White, the lesions were confined to the peritoneum. Inasmuch, however, as the lesions in these cases are similar to the more widespread lesions in the other cases, it is warrantable to group all of them in the same category as cases of more or less extensive serositis. The cases differ among themselves in the extent and intensity of the serous membrane involvement, but in all the cases the lesions are alike. We have to explain, however, the greater intensity of the lesions about the liver in the cases in which the peritoneum is the only serous membrane involved, as well as in other cases in which the pleuræ and the pericardium also are involved.

The intensity of the lesions about the liver finds its explanation in certain facts known regarding the anatomy, physiology, and pathology of the peritoneum and its circulation. The facts that interest us at present have been worked out by a number of investigators, and they have been confirmed and summarized by Clark about as follows: Fluids and solids may pass through the endothelial layer of the peritoneum, the fluids in many places, the solid particles only through the central tendon in the diaphragm. Minute particles are carried in an incredibly short time from the peritoneal cavity through the diaphragm into the mediastinal lymph vessels and glands, and thence into the blood circulation, by which they are distributed to the abdominal organs, to appear in the collecting lymph glands of these organs. The leucocytes are largely the bearers of foreign bodies from the peritoneal cavity through the diaphragm into the mediastinal lymph vessels and thence into the blood circulation. There is normally a force in the peritoneal cavity which carries the fluids and foreign particles toward the diaphragm, regardless of the posture of the patient, though gravity can greatly favor or retard the current. It is pointed out also by Clark that under normal circumstances the peritoneum can dispose of pyogenic organisms in varying number, depending upon the virulence of the organism, without producing peritonitis, and that the pathways whereby these are eliminated are as just mentioned, that is, toward the central tendon of the diaphragm and thence to the mediastinal lymph glands.

Now, what I believe is that what is true as regards experimental work and acute infections is true also as regards chronic infections; that is, the attempt on the part of the peritoneum to remove certain noxious agents—for instance, the tubercle bacillus, the infective agent of rheumatism and other diseases, probably certain toxic substances—may result in partial or complete success. In the latter instance the peritoneum may be completely rid of the infective agent, which, being carried to the mediastinal lymph vessels and lymph glands may infect the pericardium or the pleura, giving rise to a primary pleuritis or pericarditis. Subsequently the peritoneum may become infected. In other cases the attempt on the part of the peritoneum to remove the infective agent being only partially successful, the region about the liver and the under surface of the diaphragm succumbs, and a primary perihepatitis occurs. Subsequently the infective agent may travel through the diaphragm and infect the pericardium, or the pleura, or both. In these cases, on account of the peculiar lymphatic supply of the surface of the liver and the under surface of the diaphragm, the infective agent being as it were concentrated to the region about the liver, especially to the neighborhood of the suspensory ligament toward which many of the lymphatics converge, gives rise to the excessive and often hyperplastic lesions sometimes observed. The peculiarities of course and of distribution of the lymphatics of this region are also accountable for the much greater involvement of the upper surface of the liver (as contrasted with the lower surface) that is present in most of the cases. In this class of cases the primary infection of the peritoneum may be hematogenic in origin; or it may be of more local source, such as the intestinal tract or the urinary organs; or, in the case of women, it may result from some genital infection, as appears to have been the fact in Caporali's case. In a number of cases, on the other hand, a primary pericarditis occurs independently of peritoneal infection. Many of these cases are tuberculous in nature—the result generally of extension of disease from tuberculous mediastinal or peribronchial lymph glands, a subject recently discussed at length by Riesman. Following such initial infection of the pericardium the other serous membranes may become implicated in the diseased process. Thus either an ascending or a descending infection of the several serous membranes may occur; and there is no reason for not believing that in some cases all the serous membranes may become infected at the same time by the same infective agent.

The condition of the liver in these cases now demands consideration. In assigning to the liver a rôle in the production of the ascites we have to bear in mind that in 31 of the cases tabulated chronic peritonitis or chronic perihepatitis or both were present. (There is no note with regard to the condition of the liver in 1 case.) Thus in 31 of the cases the ascites may be accounted for without reference to the condition of the liver substance. In the 7 cases in which there is no note of perihepatitis or peritonitis the liver is described as nutmeg or cirrhotic, or both. In the case of cirrhosis (Feierabend) the ascites may be attributed to the cirrhosis, which may have been an independent affection or the consequence of the long-standing congestion. With regard to the remaining 6 cases—1 of Mott's showed recent inflammatory lymph in the peritoneum, Broadbent's showed recent miliary tuberculosis, whereas the reports of the other 4 cases (Mott, Cantu, Pick, and Cabot) contain no statement whatever with regard to the condition of the peritoneum.

In the absence of statements to the contrary, assuming that the peritoneum was normal in these cases, the cases in which one has to invoke the activity of the liver alone to explain the occurrence of the ascites are still very few. In the majority of cases the ascites is satisfactorily explained by the perihepatitis and the peritonitis, and this fact has to be borne in mind by those that would assign predominance to the liver in the causation of the ascites.

Again, the condition of the liver in the majority of these cases is scarcely that which we usually associate with the production of ascites. In the cases in which the liver was normal aside from compression, as well as in the cases in which there appeared to be only extension of connective tissue from the thickened capsule into the substance of the organ, predominance in the production of the ascites cannot be ascribed to the liver. Even with regard to the cases in which, in addition to the perihepatitis, cirrhosis of the liver was present, we have to bear in mind that the ascites, in most of the cases, was present at a time when clinically the liver was normal in size or enlarged and presumably not cirrhotic. With regard to the cases in which the liver was large and firm or nutmeg in appearance we have to bear in mind that most of the livers that were subjected to careful microscopic study revealed, as a rule, only varying degrees of chronic congestion and its consequences, and that in many cases the entire absence of periportal connective tissue proliferation has been emphasized. In some of the other cases the connective tissue proliferation although present has been inconspicuous—certainly in most of the cases insufficient alone to account for the ascites, a subject discussed in detail by Eisenmenger. Contrary to the opinion held by Pick and others, therefore, I believe that in the majority of cases primary importance in the production of the ascites cannot be assigned to the liver. However, that the condition of the liver may be a contributing factor in the causation of the ascites, and in the increase of an ascites produced by the serositis (perihepatitis or peritonitis), as for instance, in cases of associated cirrhosis, etc., must be conceded; that in a few cases the ascites may be brought about in the manner maintained by Pick, that is, as a direct result of the congestion of the liver, may also be conceded. These, which are rare cases, may be likened to the unusual cases of valvular disease of the heart in which disproportionate ascites occurs. Still, it is important to bear in mind that these two classes of cases differ in this respect—that an isolated ascites, such as was present for a long time in many of the cases herewith tabulated, rarely if ever occurs in valvular disease of the heart. In both disproportionate ascites is not uncommon; in valvular disease of the heart, however, the disproportionate ascites is accompanied by more or less œdema of the extremities, as well as of the trunk—an association that is not uncommon in certain cases of obliterative pericarditis. Under such circumstances the differential diagnosis between these two conditions is a matter of extreme difficulty, if not of impossibility — a subject recently discussed in detail by Türk.

At present, however, I am concerned not so much with this class of cases as with that class in which for a long time isolated ascites is the striking clinical feature. I am inclined to concur with Rosenbach, who states that in these cases the ascites must be the result of the operation of special conditions which although associated with the heart (or pericardial) disease are not the direct result of diminution of the power of the heart and of consequent congestion. While it is probable that all the cases that present the complex of obliterative pericarditis and ascites may not be of the same nature, I should like to emphasize the importance of the perihepatitis and the peritonitis in the causation of the ascites.

In this connection I cannot forbear to refer to some extremely interesting observations made some years ago by Hale White. This writer, basing his remarks upon 34 cases of cirrhosis of the liver, stated that he was not able to find the report of a single case of uncomplicated cirrhosis of the liver in which the patient long survived paracentesis of the abdomen, that not one patient lived long enough for paracentesis to become necessary a second time, and that many patients died before it became necessary at all. Referring then to a group of 10 cases that had been regarded as cases of cirrhosis of the liver but in which paracentesis had been performed more than once, he concluded: That the supervention of ascites in uncomplicated cirrhosis of the liver means that the end is near; that if a patient with ascites and cirrhosis of the liver lives long enough to require a second paracentesis, it is in the highest degree probable that either the diagnosis is incorrect or that some cause other than cirrhosis exists to explain the ascites; and that such cases nearly always turn out to be examples of chronic peritonitis, with perihepatitis, which is not a special disease but merely a part of chronic peritonitis. I cite these observations merely to emphasize the relative unimportance of the liver in the causation of an ascites that persists as long as, and necessitates as many tappings as, does the ascites in the cases at present under discussion. That recurring ascites occurs in the absence of congestive alterations in the liver is well illustrated also by the case reported by Hübler. In this case, as well as in a number of cases reported by Hale White and others, the serous membrane disease was confined to the peritoneum. Clinically these cases were the counterpart of many of the cases herewith tabulated, but at the necropsy the pleuræ and the pericardium were found intact. There was absence of congestive alterations in the liver, absence of connective tissue hyperplasia in the liver, but there were marked hyperplastic perihepatitis (zuckergussleber) and recurring ascites.

To RECAPITULATE: In a certain proportion of cases of chronic obliterative pericarditis ascites is the striking clinical symptom, and leads usually to the diagnosis of cirrhosis of the liver—whence the somewhat unhappy designation "pericarditic pseudocirrhosis of the liver," suggested by Pick, is not altogether inappropriate. Anatomically the disinguishing feature of these cases is a chronic fibrous (usually hyperplastic) inflammation of the several serous membranes of the body—a multiple serositis. In some cases the inflammation may remain localized to one or two serous membranes, but there exists a marked tendency for all the serous membranes to become involved. The several serous membranes may become involved equally or unequally; usually the lesions are most marked in the pericardium and on both sides of the diaphragm. In some cases extensive calcification of the pericardium occurs—the case herewith reported being the most marked example of this condition that I have ever encountered. In cases in which there is but partial obliteration of the pleural or the pericardial sacs there is usually also some fluid present. In many cases, however, not only is the pericardium thickened and obliterated, but it has also formed adhesions with the chest wall and the structures of the mediastinum, one or both pleuræ are thickened and obliterated, and the peritoneum is more or less thickened and adherent and contains a large quantity of fluid. In many cases the liver is adherent not only to the under surface of the diaphragm, but also to the stomach, the colon, the omentum, the anterior abdominal wall, etc. The lesions involve also the spleen, especially in those cases in which there are extensive adhesions in the left-pleura. A distinguishing feature of the lesions is the development of thick fibrous, almost cartilaginous masses of connective tissue, that encase, compress, and often distort the organs, and give rise to an appearance suggesting confectioners' icing—whence the designation "iced liver" (zuckergussleber). In many cases of primary pericarditis the liver is enlarged, smooth, and has sharp edges; later with the development of the perihepatitis, the liver lessens in size, its surface becomes somewhat irregular, its edges rounded, and sometimes the entire organ becomes much distorted; still later or in some cases earlier the appearances characterized as nutmeg, with or without atrophic alterations, may supervene. Although some slight connective tissue proliferation and some extension of newly formed connective tissue from the thickened capsule sometimes occur, the common absence of marked connective tissue proliferation in the liver is noteworthy. In some cases the lesions of cirrhosis are encountered.

As already stated, the striking clinical feature of the disease is ascites, which is present whether the pericardial and pleural changes are absent, whether they are slight, or whether they are marked. However, the interpretation of the phenomena of the disease is much facilitated by recognizing that taken as a whole—from the beginning to the end—the symptom complex is made up of symptoms due to the serositis and of symptoms due to the disturbances or failure of the circulation induced by the pericardial part of the serositis. As a matter of fact, aside from the ascites, which occurs early, and may be the only symptom for many years, the symptoms are due almost entirely, if not entirely, to failing cardiac compensation. Expressing my own opinion, I should say that there can be little doubt that presenting the complex of obliterative pericarditis and ascites, there are grouped a number of cases that differ more or less in nature the one from the other. However, I believe that in the majority of cases the ascites is due primarily to the peritonitis and the perihepatitis; that in consequence of the inflammation, the peritoneum becomes a locus minoris resistentiæ; that in consequence of the chronic pericarditis—in reality the pancarditis—the functional activity of the myocardium is interfered with; that in consequence of the lessening of the functional activity of the myocardium the circulation of the blood is more or less impeded—in some cases insensibly impeded; that the region that first manifests the sensible evidences of impeded circulation is the locus minoris resistentiæ—the peritoneum; that the relief afforded the general venous circulation by the accumulation of the ascites, probably indirectly, prevents to some extent the occurrence of œdema of the extremities; that the connective tissue hyperplasia of the liver in some of the cases is due to the same causes that induce the serous membrane inflammation—the irritant being carried by the lymphatics into the liver substance from the diseased capsule; and that in different cases the ascites is increased by the contraction of the newly formed connective tissue of the capsule, by the concomitant peritonitis in the transverse fissure of the liver should it compress the vessels (which occurs rarely if at all), by the marked congestion and its consequences, by concurrent cirrhosis (which is unusual), and by the general failure of the circulation toward the close of life.

Thus, although the disease is characterized by slow, insidious, and intermittent course, the perihepatitis and the peritonitis dominate the clinical picture of the well-developed disease. Aside from this, that is, aside from the ascites, the clinical picture varies, depending upon the point of origin of the disease. In some cases the disease is ushered in with acute pericarditis, pleuritis (which may necessitate repeated tappings), or perihepatitis (circumscribed peritonitis), which subsides and leaves the patient apparently well for a number of years. Usually, however, the lesions of the pericardium and of the pleuræ are latent, and the first sensible evidence of the disease is ascites. In some cases of primary pericarditis slight and transient œdema of the legs may be present clearly, but this is not observed in many cases, inasmuch as the pericarditis is usually latent. When œdema does occur early it usually soon subsides and does not recur until the ascites has been present for a long time or until shortly before death. Except shortly before death it is inconspicuous when contrasted with the excessive ascites. (Reference is not made in this communication to that class of cases in which general failure of the circulation occurs early and in which œdema and ascites develop pari passu.) Associated with early œdema, slight swelling of the spleen and disturbances of the gastro-intestinal tract may develop, but these usually subside with the ascites, and the disease, as a rule, is characterized by the entire absence of such symptoms until shortly before death. The ascites is characterized by the fact that it is excessive, that it necessitates repeated tappings, that it recurs rapidly after tapping, and that it may remain stationary (not necessitating tapping) for many years.

In a few cases lesions of the peritoneum appear to be entirely absent, whereas, in other cases they are so slight as to appear inadequate to cause the ascites. These cases may be likened to the similar cases of valvular disease of the heart in which disproportionate ascites is the striking clinical feature. In these cases the ascites is doubtless due directly to the congestion of the liver and its consequences, as maintained by Pick. In some cases both factors—the congestion of the liver and the peritonitis—are operative; in some cases rather the congestion of the liver, in other cases rather the peritonitis. In cases in which the congestion is the major factor we should expect more or less swelling of the spleen and disturbances of the gastro-intestinal tract; in cases in which the serositis is the major factor, we should expect an absence of such symptoms. Some distinction—anatomically, at least—may be drawn between cases in which the lesions are confined to the peritoneum and cases in which the lesions are more wide-spread. The cases in which the pericardium is unaffected reveal no congestive alterations in the liver. Clinically, however, the two classes of cases are very much alike, and the "zuckergussleber" may occur in both.

In the diagnosis of this multiple serositis or of chronic adhesive pericarditis with ascites, especial attention should be directed to the history of a previous attack of acute pericarditis, pleuritis, or perihepatitis; to the early occurrence and subsequent disappearance of the œdema of the legs; to the marked ascites, with little or no œdema of the legs; to the enlarged liver early in the course of the disease (in some cases the liver appears not to have been enlarged), and to the small and distorted, but otherwise smooth liver, in the later stages of the disease; to the absence or very late occurrence of marked enlargement of the spleen; to the tendency to the occurrence of repeated attacks of pain, tenderness, rigidity, and possibly palpable and audible frictions in the right hypochondriac region—attributable to attacks of perihepatitis; to the rapid recurrence of the ascites after tapping, and to the physical signs of adherent pericardium—without which, it may be said, the disease is incapable of diagnosis. In the diagnosis of adherent pericardium most help will be derived from: a weak or absent apex-beat, especially in cases in which there is no increase in the area of cardiac dulness; systolic retraction of a considerable area about the apex; systolic retraction of the base of the left chest posteriorly; arrest of the normal respiratory movements in the epigastric angle; imperfect descent of the apex-beat during inspiration; inadequate or entire absence of shifting of the apex-beat, with change in the posture (lateral posture) of the patient; absence of change in the limits of the cardiac dulness during the respiratory phases; absence of increase of the cardiac dulness to the right, despite marked engorgement of the veins of the neck; a diastolic shock or rebound of the heart; evidences of dilatation or hypertrophy of the heart in the absence of valvular or other disease that might cause it; absence of the characteristic changes in the heart in the presence of definite valvular disease—that is, absence of the usual hypertrophy of the right ventricle in mitral disease and of the left ventricle in aortic disease; absence of pericardial effusion in the presence of pleural and peritoneal effusions; paradoxic pulse—inspiratory diminution in the force and volume of the pulse; diastolic collapse and inspiratory swelling of the veins of the neck; and, as suggested by Musser, the non-occurrence in young subjects with valvular disease and cardiac insufficiency of the characteristic improvement following the administration of digitalis. None of these is pathognomonic of adherent pericardium; all will not be present in every case; but in many cases a sufficient number may be present to warrant the diagnosis.

Finally, this obliterative pericarditis with ascites may be distinguished from cirrhosis of the liver: by the aforementioned symptoms and signs of adherent pericardium; by the absence of the etiological factors of cirrhosis of the liver; by the slow, insidious, protracted, and intermittent course of the disease; by the long periods of standstill during which the ascites may remain stationary and the patient in good condition; by the entire absence or the transient presence of slight jaundice; by the absence in most cases of portal congestion and gastro-intestinal disturbances—hemorrhage, diarrhœa, hemorrhoids, enlargement of the spleen, marked dilatation of the superficial veins of the abdominal wall, etc.; in some cases, by the association of an enlarged, smooth, and firm liver, with marked ascites; and by the fact that in many cases the patient survives a large number of tappings.

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NOTE.—Since the foregoing paper was read the following articles have come to hand: Becker: Obliterative Pericarditis, with Hepatic Enlargement and Ascites. Philadelphia Medical Journal, March 25, 1902, vol. ix. p. 485; Simon: Zur Kenntnis der Zuckergussleber. Inaug.-Diss., Königsberg, 1900; Centralblatt für innere Medicin, 1902, vol. xxiii. p. 491; Pfannkuche: Zur Kenntnis der serösen Peritonitis und der Perihepatitis im Zusammenhang mit Perikarditis und Pleuritis. Inaug.-Diss., Kiel, 1901; Centralblatt für innere Medicin., 1902, vol. xxiii. p. 515; Strajesko: Ein Fall von Pseudocirrhose der Leber. Allgemeine Wiener medizinische Zeitung, 1902, vol. xlvii. p. 60; and Nicholls: On a Somewhat Rare Form of Chronic Inflammation of the Serous Membranes (Multiple Progressive Hyaloserositis). Studies from the Royal Victoria Hospital, Montreal, 1902, vol. i. No. 3. The article by Nicholls, which deals especially with the pathologic-anatomic features of chronic inflammation of the serous membrane, is the most exhaustive and comprehensive discussion of the subject that has yet appeared in the English language.